

March 22, 1955

Dear Dr. Demarec:

I have just mailed a parcel containing the vials, and residues of phage samples, that you had formerly provided. As I wrote you earlier, the results so far have been negative. However, we should probably try to make an exhaustive test of as many loci as can be distinguished. You did mention that you had other mutants, but if their loci are already represented in this batch, there would be little use to test them.

There are however still some gaps that leave us short of an absolutely complete test of the present material:

1). In the original shipment, the vials of "ath-B6" and "ad-D11" had lost their contents owing to leakage. These loci have not been tested as yet.

2) The lysates of hi-C30, tryp-C3 and cys-A1 had very low efficiencies of transduction of motility, so that very few swarms have so far been detected. The original supply of the lysate was exhausted before we had an opportunity to confirm the stated high titer. We would therefore be grateful for a further sample, if you can conveniently furnish it, for each of these three. In addition, in order to test the reality of the low competence, could you send us the bacterial cultures corresponding to these lysates, especially cys-A1 if not all three? In all likelihood, it is primarily a question of gradual loss of titer, but the observation should not be overlooked.

Barbara McClintock told me some more of the exciting work you have been doing on mapping sequences in linked transductions. I was especially interested to learn of the control tests, reciprocal transductions, etc. that you are carrying out to validate the system, though she did not give much detail on this. The paper that Stocker, Zinder and I published on motility transduction (JGM) gave only a hint of the largely fruitless efforts we expended on mapping of the linked Fla-H<sub>1</sub> system. This was partly hindered by the phenotypic interaction of these loci, and consequent confusion, or insusceptibility of selection, of certain combinations. But just as discouraging was the fact that the apparent segregation ratios of a linked, unselected marker varied greatly depending on the exact identities of the recipient and donor strains. This may be closely connected with the fact that our markers here derived from a wide variety of strains and serotypes (*S. typhimurium*, paratyphi B, heidelberg, etc.), while yours are doubtless more unitary changes in a uniform genotypic background. At any rate, my conclusion has been that we could not reliably map the Fla-H system in a linear sequence with the available technique, although we have made any number of speculative attempts.

Yours sincerely,

  
Joshua Lederberg